

This is another argument against the probability of these being micro-organisms. We cannot suppose the comatose form of malarial poisoning to be a special disease, and were a lower organism found in this, we should also find it in other cases.

We present here the facts only as we have found them; we confess our inability to say what these hyaline bodies are. That they have been seen before by other observers appears most likely.

We hope that the paper will direct attention to the subject in localities where this form of malarial poisoning is more common, and possibly with more material at command, and with more skilful observation, the question will be satisfactorily solved.

ARTICLE X.

OBSERVATIONS ON THE CUTANEOUS AND DEEP REFLEXES. By PHILIP COOMBS KNAPP, A.M., M.D. (Harvard), of Boston.

THE following notes upon the cutaneous and deep reflexes are taken from a series of observations upon 239 persons admitted to the Nervous and Renal Service at the Boston City Hospital, during the latter half of the year 1883. The points noted were the presence or absence of the different reflexes in each patient, a comparison of the reflexes upon the two sides, and a rough estimate of the degree of contraction after the irritation. All the reflexes were tested upon the bare skin, some sharp-pointed instrument—either an æsthesiometer or the point of a pen-knife—being used for the cutaneous reflexes, and a rubber-headed percussion hammer for the deep reflexes.

The cutaneous reflexes examined were the plantar, cremaster, gluteal, abdominal, epigastric, erector spinæ, and scapular.¹ Of these, the plantar reflex was the most constant. In 234 cases it was absent in 17, 10 of which had some direct lesion of the reflex arc, either of the peripheral nerves or of the lumbar cord, six having multiple neuritis, two myelitis affecting the lumbar cord, and two locomotor ataxia. Of the remaining seven cases, three were comatose, two from alcohol, and one from chronic meningitis. The other four will be briefly described as follows:—

CASE I.—Michael M., 49; alcohol to excess; no definite symptoms to be obtained; much mental impairment; general pains and questionable girdle sensation; no evidence of paralysis. In a short time became comatose and died.

CASE II.—Peter M., 58; attacks of vertigo; later, left hemiplegia with contracture; marked mental impairment. In a few weeks became gradually comatose and died. Tumor in corpus callosum.

¹ A description of these reflexes and the method of testing them may be found in W. R. Gowers' *Diagnosis of Diseases of the Spinal Cord*, ed. 1884, pp. 17, 18.

TABLE I.—Cases showing Absence of Plantar Reflex.

N. Normal. o. Absent. N.T. Not Tested. + Increased. — Diminished. r. Right. l. Left.
The numbers are those of the whole series of 239 cases.

Number of observation.	Name.	Disease.	Cutaneous Reflexes.						Deep Reflexes.						
			Cremaster.	Genital.	Abdominal.	Epi-gastric.	Erector spinae.	Scapular.	Patellar.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrist.
8	Mary F.	Multiple neuritis .	N.T.	N.T.	o.	o.	N.T.	N.T.	o.	o.	o.	o.	o.	o.	o.
9	John C.	" .	N.	N.T.	o.	o.	N.T.	N.T.	o.	o.	N. T.	o.	o.	o.	N.T.
16	Edward M.	" .	o.	N.T.	o.	o.	N.T.	N.T.	o.	o.	o.	o.	o.	o.	N.T.
44	Kate C.	" .	N.T.	—	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	N.T.
92	Thomas G.	" .	o.	N.	N.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
190	Mary N.	" .	N.T.	o.	o.	o.	—	o.	o.	o.	o.	o.	o.	o.	o.
143	Abbie K.	Myelitis .	N.T.	N.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
228	Morsbury J.	" .	o.	N.	o.	N.	o.	o.	o.	o.	o.	N.	o.	o.	N.
119	George T.	Locomotor ataxia .	o.	o.	o.	o.	o.	o.	o.	o.	o.	—	o.	o.	o.
200	Joseph M.	" .	N.	o.	N.	N.	N.	o.	o.	o.	o.	much	o.	o.	o.
179	Timothy S.	Alcoholic coma .	o.	o.	o.	o.	o.	o.	o.	o.	o.	—	o.	o.	o.
181	Edward H.	" .	o.	N.T.	o.	o.	N.T.	N.T.	o.	o.	o.	—	o.	o.	N.T.
149	Mary S.	Chronic meningitis [left hemiplegia].	N.T.	N.T.	o.	o.	N.T.	N.T.	N.	o.	o.	N.	R. — L.O.	o.	N.T.
57	Michael M.	Meningitis ? .	o.	o.	o.	o.	o.	o.	o.	o.	o.	—	R.O. L.N.	o.	o.
71	Peter M.	Cerebral tumor [left hemiplegia]	o.	o.	o.	o.	o.	R. — L.O.	R. — L.N.	o.	o.	o.	o.	o.	N.
65	John R.	Alcoholism .	N.	o.	o.	o.	o.	o.	N.	o.	o.	N.	o.	o.	o.
102	Sarah M.	Mania, nephritis .	N.T.	o.	o.	o.	o.	o.	o.	o.	o.	—	o.	o.	o.

CASE III.—John R., 39; alcohol to excess and exposure to sun. Headache, vertigo, tremor, loss of appetite, insomnia.

CASE IV.—Sarah M., 49; vomiting, headache, slight œdema of feet before entrance. Excited, noisy, and incoherent; refused food; delusions of poisoning. Urine, trace of albumen, hyaline and granular casts. (See Table I.)

In these seven cases, then, there is no clear evidence of a lesion which might act as a direct break in the path of the transmission of the nerve currents, but there is evidence of disturbance of the higher nerve centres. It is a well-known fact that a lesion in one cerebral hemisphere, as a hemorrhage, causing hemiplegia on the opposite side of the body, may, at least temporarily, diminish or abolish the cutaneous reflexes on the paralyzed side, showing that the cerebral disturbance has a direct inhibitory influence. This seems a satisfactory explanation of the absence of the reflex in the first five cases; in the last two the disturbance seems hardly severe enough to have much inhibitory action, yet such an explanation is not impossible, and is the only one I can suggest. In no case, at all events, was it absent where there was not some well-marked disturbance of the nervous system, either a direct lesion of the reflex arc or some cerebral disorder.

The cremaster was second in constancy among the cutaneous reflexes. In 21 cases out of 167 it was absent, and in two cases the cause of its absence was local—extreme œdema of the scrotum, and double hernia with hydrocele. Eight of the other 19 cases had the plantar reflex also absent—two cases of multiple neuritis, one of locomotor ataxia, one of acute myelitis, one of tumor of the brain (Case II.), two of coma, and one of obscure cerebral disease (Case I.). Of the other eleven, one had multiple neuritis, one chronic myelitis, a third meningitis with obscure spinal symptoms and paraparesis, a fourth, with a history of convulsions, had clonic spasms and loss of power in one leg, with some tenderness along the nerve trunks, the fifth was a recent hemiplegia, the sixth was an alcoholic case, delirious, with occasional convulsions later, who had chronic interstitial hepatitis and nephritis, one had uræmic convulsions, three were comatose (two from uræmia and one from alcohol), and the last, an alcoholic subject, had acute pneumonia with slight delirium. (See Table II.)

These cases, then, with a single exception, show either a direct lesion of the reflex arc, or severe disturbance of the higher nerve centres which might inhibit the reflex, even more clearly than the cases where the plantar reflex was absent. The delirium in the case of pneumonia was slight, and does not seem sufficient to explain the absence of the cremaster reflex in that case, yet it was the only nervous disturbance to account for it.

Following the analogy of those cases of hemiplegia which show the cutaneous reflexes absent on the paralyzed side, the absence of the

TABLE II.—Cases showing Absence of Cremaster Reflex.

Number of observation.	Name.	Disease.	Cutaneous Reflexes.						Deep Reflexes.								
			Plantar.	Gluteal.	Abdominal.	Epigastric.	Director spine.	Scapular.	Patellar.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrist.	Costal.	Lumbar fascia.
85	Owen M.	Chronic nephritis [oedema scrotum]	N.	N.	—	o.	o.	o.	o.	o.	—	o.	o.	o.	—	o.	o.
195	John K.	Epilepsy [hernia, etc.]	N.	N.T.	—	o.	o.	N.T.	o.	o.	—	o.	o.	o.	o.	N.T.	N.T.
16	Edward M.	Multiple neuritis.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
92	Thomas G.	"	o.	N.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
119	George T.	Locomotor ataxia	o.	—	o.	o.	o.	o.	o.	o.	N.	o.	o.	o.	o.	o.	o.
228	Morsbury J.	Myelitis	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
71	Peter M.	Cerebral tumor [left hemiplegia]	o.	o.	o.	o.	o.	R.—	o.	o.	R.—	o.	o.	o.	R.O.	o.	N.
173	Timothy S.	Alcoholic coma	o.	o.	o.	o.	o.	L.O.	o.	o.	L.N.	o.	o.	o.	L.N.	o.	o.
181	Edward H.	"	o.	N.T.	o.	o.	o.	N.T.	o.	o.	o.	o.	o.	o.	N.T.	N.T.	N.T.
57	Michael M.	Meningitis?	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
112	Horace W.	Multiple neuritis	N.	N.	o.	o.	o.	o.	o.	o.	N.	o.	o.	o.	o.	o.	o.
175	Horace A.	Combined sclerosis [chronic myelitis]	N.	o.	o.	o.	o.	o.	+	Slight clonus	N.	R.N. R.— L.O. L.O.	+	o.	o.	o.	o.
153	William S.	Meningitis?	N.	o.	o.	o.	o.	N.	o.	o.	N.	N.	o.	o.	N.	N.	N.
115	John B.	Epilepsy?	N.	o.	o.	o.	—	o.	o.	o.	N.	—	o.	o.	N.	o.	o.
230	Robert R.	Right hemiplegia	N.	N.T.	o.	o.	N.T.	N.T.	R.—	o.	o.	o.	o.	o.	N.	N.T.	N.T.
192	Dwight S.	Alcoholism, etc.	N.	N.	o.	o.	o.	o.	o.	o.	N.	o.	o.	o.	N.T.	o.	o.
70	John E.	Uremic convulsions [during attack].	N.	N.T.	o.	o.	N.T.	N.T.	+	Pre- sent	o.	o.	o.	o.	o.	N.T.	N.T.
73	Michael M.	Uremia [delirious]	N.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.	o.
96	Calvin A.	Alcoholic coma	—	o.	o.	o.	N.	o.	o.	o.	—	o.	o.	o.	o.	o.	o.
189	Michael P.	Uremic coma	N.	N.T.	o.	o.	N.T.	N.T.	o.	o.	o.	o.	o.	o.	N.	N.	N.T.
191	Michael A.	Acute pneumonia	N.	o.	o.	o.	N.	o.	N.	o.	N.	o.	o.	o.	N.	o.	o.

plantar and cremaster reflexes in so many cases of cerebral disease of one sort or another, where the cord and peripheral nerves were intact, gives additional support to the hypothesis that an irritative lesion in the brain, exciting the inhibitory centres in the basal ganglia and the motor fibres in the pyramidal tract, may inhibit the action of the cells in the gray matter in the cord, and so abolish reflex action.

Although the absence of these reflexes may be thus explained, the observations do not permit the opposite course of reasoning, that, having a lesion in the cord or peripheral nerves, or an irritative lesion in the brain, these reflexes must be absent. Their persistence in locomotor ataxia has long been noticed, and has been explained by Bramwell by the theory that nerve-fibres conducting sensations from the skin pass directly into the posterior cornua, while fibres from the deeper parts, like the tendons, pass through the posterior root zones.¹ The plantar reflex was present in six out of eight cases of locomotor ataxia, the cremaster in seven, the eighth case being a woman. The plantar reflex was seen in two cases of coma where the cremaster was absent, and in nine out of fourteen cases of multiple neuritis; the cremaster was seen in five cases out of eight of the same disease. Their presence, therefore, does not enable one to exclude such diseases, but their absence usually, if not always, implies some nervous disturbance.

The other cutaneous reflexes appeared to be of much less value. The gluteal reflex was third in constancy, but in 179 cases it was absent in 66, and these differed so much as to render deductions from its absence of very little worth. Many of the cases, from the class of diseases admitted to the service, had some nervous trouble, but its absence was seen, not only in hemiplegia, locomotor ataxia, and multiple neuritis, but also in various neuralgias and the localized paralyses of the upper extremity from injury of the circumflex or musculo-spiral nerve, and even in cases of renal or cardiac disease and phthisis, so that it would be unsafe to make any assertions as to the cause of its absence.

The same thing may be said of the abdominal reflex, which was absent in 97 cases out of 239, cases of as varied sorts as those just mentioned.

The result of testing the epigastric reflex, which Gowers claims is singularly uniform, was still more unsatisfactory, for it was absent in more than half the cases, namely, in 142 out of 239.

The cutaneous reflexes of the back, the erector spinæ, and the scapular were of such rare occurrence that the question naturally rose whether their presence might not be pathological. The erector spinæ reflex was present in 45 cases out of 178, but among these cases, besides various nervous diseases, were acute nephritis, hæmatemesis, acute rheumatism, debility, and pneumonia. The scapular reflex was present but 15 times in 177 cases, and these 15 cases again varied so much that no inferences

¹ B. Bramwell, *Diseases of the Spinal Cord*, ed. 1882, p. 111, note.

could be drawn from its presence. One point of some interest, however, was noted in regard to those reflexes, namely, some relation between their presence and that of the corresponding deep reflexes. In 33 cases out of 178 the reflex from the lumbar fascia was present; in 17 of these cases the erector spinæ reflex was also found. In 45 cases out of 178 there was a reflex from the spine of the scapula, and in ten of these the scapular cutaneous reflex was present.

The observations upon the increase or diminution of the cutaneous reflexes seemed to indicate that such a variation was of only moderate value in diagnosis. Such tests as are employed at the bedside are necessarily rough, and the normal standard is only an approximate one which varies with each observer, and variations from this standard cannot be measured with any accuracy. Again, the reflex may vary in intensity in healthy individuals, a slight plantar reflex being no more pathological than a slight patellar reflex, and the intensity is modified by several conditions, like thickness of the plantar epidermis, natural susceptibility to tickling, and even expectant attention. I have repeatedly seen an unexpected prick of the sole of the foot followed by a vigorous contraction of the whole leg, while subsequent pricks gave merely a moderate drawing up of the leg or only a contraction of the toes. A diminution of the reflexes is a thing to be expected in neuritis, in myelitis, or in hemiplegia, and often gives useful information, but an equal feebleness of contraction may be seen in nephritis, dyspepsia, valvular disease of the heart, lumbago, anæmia, phthisis, and pleurisy, so that its value depends upon its relation to other symptoms. Increase of the cutaneous reflexes, too, has not the significance of increase of the deep reflexes, for it was noted in cases of paraplegia and neuritis where the patellar reflex was absent, as well as in myelitis and multiple sclerosis, involving the lateral columns, where the deep reflexes were exaggerated, and also in cases where there was no nervous trouble at all, as in phthisis, nephritis, hepatitis, and in health. As an isolated symptom neither diminution nor increase of the cutaneous reflexes has any significance, but with other symptoms, and especially by comparing the different reflexes with each other, it often gives much information.

If the reflexes differ on the two sides, however, their value in diagnosis is much greater. The plantar reflex differed in ten cases, four of which had hemiplegia, one hemianæsthesia, and one unilateral epilepsy, the reflex being present on the convulsed side and absent on the other. In a case of uræmic convulsions the reflex was absent on one side, but the convulsions were general. It was diminished on one side in a case of reflex paraplegia following cystitis, and in another obscure case of paraplegia, while a similar difference was noted in a case of melancholia with excitement, where the patient's restlessness rendered the examination somewhat untrustworthy.

The cremaster reflex differed in but two cases, one the case of uni-

lateral epilepsy just mentioned, the other an obscure case of spinal disease, probably an irregular form of locomotor ataxia.

The gluteal differed in three cases of hemiplegia, the case of unilateral epilepsy, and a case of chronic myelitis, where the deep reflexes were exaggerated on the side where the gluteal was present.

The cases in which the abdominal and epigastric differed were not all so clearly cases where a unilateral affection could be made out. The abdominal reflex differed in seven cases: one, hemiplegia, the second, an irregular case of locomotor ataxia, the third, unilateral epilepsy, the fourth, left hemiparesis following a fall on the head, where the abdominal and epigastric reflexes were absent on the paretic side, the fifth, cerebral syphilis, with these reflexes increased on one side and the deep reflexes on the other, the sixth, dementia with lead found in the urine, and the seventh, chronic parenchymatous nephritis.

The epigastric reflex differed in five cases, three of which have just been mentioned, hemiparesis, cerebral syphilis, and dementia; the fourth had hemiplegia; the fifth had epilepsy, and was examined after the convulsion was over.

The erector spinæ reflex differed in two cases, multiple sclerosis and hemiplegia.

The scapular reflex differed in three, two hemiplegias and the case of multiple sclerosis, which had marked unilateral symptoms.

In nearly every case, therefore, there was evidence of disease of the brain or cord, either unilateral or more marked upon one side, the chief exceptions being in the cases where the abdominal and epigastric reflexes differed. In one or two cases, notably the case of nephritis, careful examination could not discover a reason for the difference; hence I am not prepared to assert that such a difference is always pathological, but I believe that it is usually a sign of some unilateral disturbance of the nervous system, and that it always demands careful investigation. (See Table III.)

Of much greater interest are the contractions obtained by percussion of tendons, periosteum, and fasciæ—the deep reflexes. Of these, the following were tested: the reflexes from the patellar and triceps tendons and the extensor tendons of the wrist, and the clonuses of the ankle, wrist, and toe; the periosteal reflexes from the tibia, radius, ulna, third costal cartilage, and spine of the scapula; and the reflex from the lumbar fascia. In a few cases where the other tendon reflexes were exaggerated the patellar clonus and the front tap contraction were tested.¹

In all examinations of the deep reflexes that from the patellar tendon from its constancy in health, its value in diagnosis, and the study which has been made of it, is of the first interest. In 47 cases out of 239 this reflex was absent on both sides. It is admitted that in lesions of the

¹ For a full discussion of these reflexes, see J. Ross, *Diseases of the Nervous System*, ed. 1882, vol. i. pp. 140–154, and W. R. Gowers, *op. cit.*, pp. 19–34.

TABLE III.—Cases in which the Reflexes differed.

In this table + and — under ankle clonus and the less frequent reflexes, refer to the intensity of the contraction rather than to any deviation from an imaginary normal standard.

Number of observation.	Name.	Disease.	Cutaneous Reflexes.							Deep Reflexes.								
			Plantar.	Cremaster.	Gluteal.	Abdominal.	Ergastic.	Erector spine.	Scapular.	Patellar.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrists.	Costal.	Lumbar fascia.
2	Richard L.	Myelitis	+	—	o.	o.	o.	o.	o.	+	R.— L.O.	o.	+	R.— L.N.	R.— L.O.	+	o.	N.
4	Ira H.	Paralysis agitans [left side affected] .	N.	N.	N.T.	o.	o.	N.T.	N.T.	N.	o.	o.	+	R.— L.N.	o.	R.N.	N.T.	N.T.
5	Martin Q.	Locomotor ataxia	N.	N.	N.T.	—	—	N.T.	N.T.	R.— L.O.	o.	o.	o.	o.	o.	—	N.T.	N.T.
13	Lizzie F.	R. hemianesthesia [hysterical] . .	R.O. L.—	N.T.	N.T.	N.	N.	N.T.	N.T.	N.	o.	o.	o.	o.	o.	o.	N.T.	N.T.
13	Lizzie F.	Right hemiplegia and hemianesthesia	R.O. L.—	N.T.	R.O. L.—	N.	N.	N.T.	N.T.	R.N. L.—	o.	o.	—	R.— L.O.	o.	o.	o.	o.
36	Edward A.	R. hemiplegia	N.	N.	N.T.	N.	N.	N.T.	N.T.	N.	o.	o.	o.	R.— L.O.	o.	N.	N.T.	N.T.
37	Owen K.	Epilepsy [recovery]	N.	N.	—	N.	R.N. L.O.	o.	o.	—	o.	o.	N.	o.	o.	R.O.	o.	N.
40	Patrick D.	Spinal concussion	N.	N.	N.T.	N.	N.	o.	N.T.	R.N. L.—	o.	N.T.	N.	N.	—	—	o.	o.
42	Timothy M.	Right hemiplegia	N.	N.	N.T.	—	o.	N.T.	N.T.	N.	o.	o.	N.	N.	o.	—	N.T.	N.T.
45	John F.	Hypochondriasis	N.	N.	N.T.	N.	o.	—	o.	R.— L.N.	o.	o.	N.	N.	—	o.	o.	o.
71	Peter M.	Cerebral tumor [left hemiplegia] .	o.	o.	o.	o.	o.	o.	o.	R.— L.O.	o.	R.— L.N.	R.— L.O.	o.	o.	R.O.	o.	N.
72	Michael G.	Sciatica, right	N.	N.	o.	N.	o.	o.	o.	R.— L.N.	o.	o.	—	o.	o.	N.	o.	o.
73	Michael M.	Uræmic convulsions	R.N. L.O.	—	N.T.	o.	o.	N.T.	N.T.	o.	o.	o.	—	o.	o.	—	N.T.	N.T.
97	Cornelius C.	Ch. paren. nephritis	N.	+	+	R.N. L.O.	o.	o.	o.	—	o.	o.	N.	N.	—	N.	o.	N.
99	Bart H.	R. unilat. epilepsy [during fits] . .	R.N. L.O.	R.N. L.O.	R.N. L.O.	R.— L.O.	o.	o.	o.	+	R.— L.O.	+	+	+	+	N.	R.— L.N.	N.

TABLE III.—Continued.

Number of observation.	Name.	Disease.	Cutaneous Reflexes.							Deep Reflexes.						
			Plantar.	Cremaster.	Gluteal.	Abdominal.	Epigastric.	Erector spinae.	Scapular.							
99	Barth H.	Recovery	N.	N.	N.	N.	R.— L.N.	O.	O.	Patellar.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrists.
107	Edwin M.	Multiple sclerosis [1 ft side most affected]	+	N.	O.	N.	N.	R.— L.—	R.— L.—	+	+	+	N.	R.— L.—	R.— L.—	O.
109	John T.	Sciatica, right	N.	N.	N.	N.	N.	—	O.	R.— L.—	O.	O.	N.	—	O.	O.
127	Patrick O.	Meningitis ?	N.	N.	N.	much	much	O.	O.	R.— L.—	O.	O.	N.	O.	O.	O.
130	Martin C.	Sciatica, right	—	N.	O.	O.	O.	—	O.	R.— L.—	O.	O.	N.	O.	O.	O.
133	Cyrus C.	Cerebral concussion, left hemiparesis .	N.	N.	O.	R.N. L.O.	R.— L.O.	O.	O.	N.	O.	O.	N.	—	O.	O.
134	Mary D.	Cranial neuralgia, left	N.	N.T.	N.	O.	O.	O.	O.	R.N. L.O.	O.	O.	—	N.	O.	N.
145	Cornelius F.	L. hemiplegia [embolism]	R.N. L.—	N.	O.	O.	O.	R.N. L.O.	O.	O.	O.	O.	N.	—	O.	O.
148	Theodore B.	Reflex paraplegia	R.N. L.—	N.	N.	O.	O.	O.	O.	slight +	O.	O.	N.	—	O.	O.
149	Mary S.	Ch. meningitis, left hemiplegia	O.	N.T.	N.T.	O.	O.	N.T.	N.T.	N.	O.	O.	N.	R.— L.O.	O.	R.N. L.O.
150	William C.	Herpes Zoster, right	N.	N.	N.	N.	O.	O.	O.	N.	O.	O.	R.O. L.N.	O.	O.	O.
153	Annie M.	Paraplegia	N.	N.T.	N.	O.	O.	O.	O.	O.	O.	O.	R.O. L.—	—	O.	O.
158	Otto W.	L. hemiplegia	R.— L.O.	N.	R.N. L.O.	R.N. L.O.	O.	O.	O.	N.	O.	O.	N.	N.	R.N. L.O.	O.
161	John H.	Chronic myelitis	N.	N.	N.	N.	N.	N.	N.	+	R.— L.O.	O.	N.	N.	N.	N.
164	Edward H.	Diabetes mellitus	—	N.	N.	N.	O.	O.	O.	R.— L.O.	O.	O.	N.	N.	O.	O.

TABLE III.—*Concluded.*

Number of observation.	Name.	Disease.	Cutaneous Reflexes.						Deep Reflexes.										
			Plantar.	Cremaster.	Gluteal.	Abdominal.	Epiclas- tric.	Erector spine.	Scapular.	Patellar.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrists.	Costal.	Lumbar fascia.	Spine of scapula.
166	Mary O.	Paraplegia ?	R.N. L.—	N.T.	O.	O.	O.	O.	O.	R.N. L.—	O.	O.	N.	R.N. L.—	O.	O.	O.	O.	N.
168	William E.	R. hemiplegia	R.N.	N.	O.	N.	R.O. L.—	O.	O.	N.	R.— L.O.	R.— L.O.	R.— L.O.	R.N. L.O.	R.N. L.O.	R.N. L.O.	—	O.	O.
173	Thomas C.	Multiple neuritis	+	—	—	N.	N.	N.	N.	O.	O.	O.	O.	R.— L.O.	O.	N.	O.	O.	N.
175	Horace A.	Combined sclerosis [chronic myelitis]	N.	O.	O.	O.	O.	O.	O.	+	—	+	N.	R.N. L.O.	R.— L.O.	O.	N.	O.	O.
176	John L.	Chronic nephritis	N.	N.	N.	O.	O.	O.	O.	N.	O.	O.	O.	N.	N.	R.O. L.—	—	—	—
185	Mary O.	Melancholia	R.— L.N.	N.T.	N.	N.	—	—	N.	O.	N.	O.	O.	O.	O.	O.	O.	O.	O.
186	Nathan W.	Cerebral syphilis	—	N.	N.	R.N. L.—	R.N. L.—	O.	O.	R.— L.N.	R.— L.O.	O.	—	R.— L.O.	O.	O.	N.	O.	O.
193	Julia H.	Dementia, left hemiplegia ?	—	N.T.	O.	O.	O.	O.	O.	O.	O.	O.	—	—	O.	O.	R.N. L.O.	O.	O.
194	Michael F.	Combined sclerosis [chronic myelitis]	—	N.	R.O. L.N.	O.	O.	O.	O.	+	R.— L.—	+	N.	O.	O.	O.	O.	O.	O.
201	Thomas M.	Multiple neuritis	N.	N.	O.	N.	N.	N.	N.	O.	R.— L.O.	O.	—	O.	O.	O.	N.T.	O.	O.
212	Bridget G.	L. hemiplegia	N.	N.T.	R.N. L.O.	O.	O.	O.	O.	R.— L.O.	O.	O.	N.	N.	O.	O.	O.	O.	O.
221	John M.	Dementia [lead poison]	N.	N.	O.	R.O. L.N.	R.O. L.—	O.	O.	R.O. L.N.	O.	O.	N.	N.	O.	O.	N.	O.	O.
222	Annie C.	Anæmia	N.	N.T.	N.	O.	O.	O.	O.	N.	O.	O.	N.	R.— L.O.	R.— L.O.	O.	—	O.	O.
224	Mary C.	Apoplexy	N.	N.T.	N.T.	O.	O.	N.T.	N.T.	R.— L.N.	O.	O.	N.	O.	O.	O.	N.	N.	N.T.
230	Robert R.	Right hemiplegia	N.	O.	N.T.	O.	O.	N.T.	N.T.	R.— L.N.	O.	O.	N.	O.	O.	O.	N.	N.T.	N.T.
235	Charles M.	Locomotor ataxia	—	R.O. L.—	N.	R.— L.N.	O.	O.	O.	O.	O.	O.	N.	N.	O.	O.	N.	O.	N.

reflex arc, as in neuritis, meningitis involving the nerve-roots, locomotor ataxia, myelitis, anterior poliomyelitis, progressive muscular atrophy, and pseudo-hypertrophic muscular paralysis, the patellar reflex may be absent. Hence thirteen cases of multiple neuritis, seven cases of locomotor ataxia, two cases of myelitis, two cases of meningitis, a case of diphtheritic paraplegia, and three cases of paraplegia, or paraparesis of obscure spinal origin, in all of which the patellar reflex was absent, may be dismissed without further comment. In very stout people with short tendons Westphal states¹ that it is very difficult to get the reflex; the difficulty is increased if the legs are œdematous. This may account for the failure to obtain it in three very stout women. Four more were cases of coma, one from uræmia, three from alcohol; in one of these the reflex was well marked on recovery the next day; the others were not tested. The remaining twelve will be briefly described:—

CASE I.—John F., 48. Intercostal neuralgia, probably malarial. Hole in skull, from bullet wound, over second frontal convolution, right. At times convulsive movements of right arm, vertigo, loss of consciousness.

CASE II.—Michael M., 49. Alcohol to excess. No definite symptoms to be obtained; much mental impairment; general pains and questionable girdle sensation; no evidence of paralysis. In a short time became comatose and died. [Case I. under plantar reflex.]

CASE III.—Edward R., 44. Alcohol to excess. Nervous, tremulous, sleepless. Œdema of legs. Albuminuria.

CASE IV.—Terence M., 65. Alcohol to excess. Delirious two weeks. Much mental impairment; gives no intelligent history; complains chiefly of chest. Ocular paresis; hallucinations of sight. Later coma and death.

CASE V.—Michael M., 30. Alcohol to excess. General epileptiform convulsions. No œdema; urine of acute nephritis. Later delirious, hallucinations of vision; coma and death. No reflex in convulsive or delirious stage.

CASE VI.—Dwight S., 35. Alcohol to excess. Delirious and tremulous. Occasional epileptiform convulsion. Albumen, hyaline and granular casts. Liver much enlarged. Reflex not tested in convulsion.

CASE VII.—Cornelius F., 32. Disease of aortic valves. Right hemiplegia 6–7 months before; recovery. Left hemiplegia.

CASE VIII.—Julia H., 60? Much demented. Question of old left hemiplegia. Slight paresis of left side of face.

CASE IX.—Sarah M., 49. Vomiting, headache, slight œdema of feet before entrance. Excited, noisy, and incoherent; refused food; delusions of poisoning. Trace of albumen, hyaline and granular casts. [Case IV. under plantar reflex.]

CASE X.—Jeremiah B., 44. Fell from ladder, striking left hip. Much pain about hip. No evidence of fracture. No pain down leg.

CASE XI.—Ellen W., 35. Facial neuralgia; cancer of breast; systolic murmur at apex of heart. Badly nourished, broken-down, neuropathic subject.

CASE XII.—Michael S., 11. Question of previous scarlatina. Mild acute nephritis, some œdema. No nervous symptoms. (See Table IV.)

¹ C. Westphal, Ueber das Verschwinden und die Localisation des Kniephänomens. Berlin. Klinisch. Wochenschrift, Jan. 3, 10, 1881.

TABLE IV.—*Concluded.*

Number of Observation.	Name.	Disease.	Cutaneous Reflexes.						Deep Reflexes.								
			Plantar.	Cremas- ter.	Gluteal.	Abdom- inal.	Epiplas- mic.	Erector spine.	Scapular.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrist.	Costal.	Lumbar fascia.
207	Daniel G.	Diphtheritic paraplegia	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
6	Charles G.	Paraplegia	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
83	Thomas C.	"	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.
152	Annie M.	"	N.	N.T.	N.	N.	N.	N.	N.	N.	N.	N.	N.	N.	N.	N.T.	N.
63	Julia P.	Hypertrophic cirrhosis [very stout]	+	N.T.	N.T.	O.	O.	O.	N.T.	N.T.	O.	O.	O.	O.	O.	N.T.	N.T.
141	Ann M.	Chronic nephritis [stout, oedema of legs]	N.	N.T.	N.	N.	N.	N.	O.	O.	O.	O.	O.	O.	O.	O.	O.
234	Ellen R.	Dyspepsia [very stout]	N.	N.T.	N.	N.	N.	N.	O.	O.	N.	O.	O.	O.	O.	O.	O.
96	Calvin A.	"	—	O.	O.	O.	O.	O.	N.	O.	N.	O.	O.	N.	O.	O.	O.
179	Timothy S.	"	—	O.	O.	O.	O.	O.	O.	O.	O.	O.	O.	O.	N.T.	O.	O.
181	Edward H.	"	O.	O.	N.T.	O.	O.	N.T.	N.T.	O.	O.	O.	O.	O.	O.	N.T.	N.T.
189	Michael P.	Uremic coma	N.	O.	N.T.	O.	O.	N.T.	N.T.	O.	O.	O.	O.	O.	O.	N.T.	N.T.
19	John F.	Intercostal neuralgia	N.	N.	N.T.	O.	O.	N.T.	N.T.	O.	O.	O.	O.	O.	N.	N.T.	N.T.
57	Michael M.	Meningitis?	N.	N.	N.T.	O.	O.	N.T.	N.T.	O.	O.	O.	O.	O.	N.	N.T.	N.T.
64	Edward R.	Alcoholism	N.	—	N.	O.	O.	N.	O.	O.	O.	O.	O.	O.	N.	O.	O.
81	Terence M.	Meningitis?	N.	N.	N.	X.	X.	N.	O.	O.	O.	O.	O.	O.	O.	O.	O.
73	Michael M.	Uremic convulsions	R.N.	—	N.T.	O.	O.	O.	N.T.	N.T.	O.	O.	O.	O.	—	N.T.	N.T.
73	Michael M.	Uremia [delirious]	L.O.	O.	N.T.	O.	O.	O.	O.	O.	O.	O.	O.	O.	—	O.	O.
192	Dwight S.	Alcoholism, etc.	N.	O.	N.	O.	O.	O.	O.	O.	N.	O.	O.	O.	O.	O.	O.
145	Cornelius F.	L. hemiplegia	R.N.	N.	O.	O.	O.	O.	R.N.	O.	N.	—	O.	O.	—	O.	O.
193	Julia II.	Dementia, left hemiplegia?	L.—	N.T.	O.	O.	O.	O.	O.	O.	—	—	O.	O.	R.N.	O.	O.
102	Sarah M.	Mania, nephritis	O.	N.T.	O.	N.	N.	O.	O.	O.	—	—	O.	O.	L.O.	O.	O.
20	Jeremiah B.	Spinal concussion?	—	N.	N.T.	N.	N.	N.T.	N.T.	O.	—	—	O.	O.	—	N.T.	N.T.
136	Ellen W.	Facial neuralgia, etc.	N.	N.T.	O.	N.	N.	O.	O.	O.	—	—	O.	O.	—	O.	O.
47	Michael S.	Acute nephritis	N.	—	N.	N.	N.	N.	O.	O.	N.	O.	O.	O.	N.	O.	O.

Finding the patellar reflex absent in every case of coma that was examined, and being led to believe that in one case at least its absence was due to the coma, I naturally was brought to the inquiry whether etherization affected the reflex. The only statement upon the point that I found was that by Horsley that ether and nitrous oxide do not diminish it.¹ I took occasion to test the reflex of a patient that I had etherized to remove a chalazion—a nervous, anæmic man with a well-marked reflex before taking ether—and found that when he was so far etherized that his conjunctival reflex was gone and his limbs were relaxed, the reflex was absent. Being anxious to make further investigations, by the kindness of Dr. Post and the visiting surgeons of the Boston City Hospital, I was permitted to test the patellar reflex in a number of patients before and during etherization. A satisfactory test was made on thirteen adults and four children. In every case the reflex was well marked before etherization, and the second test was made when the conjunctival reflex was lost and the limbs were thoroughly relaxed. In one case there was a very slight increase, in nine cases there was no change, in two cases it was somewhat diminished, and in five, one adult and all the children, it was entirely absent.

Before discussing the cause of the absence of the patellar reflex I will speak of the other changes found in it and of the other deep reflexes of the lower extremity. A diminution of the contraction, although often accompanying some definite lesion of the reflex arc, as in incipient locomotor ataxia or in mild cases of multiple neuritis, is not of much weight, for a very feeble contraction is not inconsistent with perfect health. Absence or marked exaggeration is alone of significance.²

In eight cases the patellar reflex was markedly increased, and in seven it proved a most valuable symptom. Four of the cases had myelitis, one an acute myelitis in the dorsal region, the other three chronic myelitis, probably combined sclerosis of the posterior and lateral columns; one was a case of multiple sclerosis involving the lateral columns; one a case of secondary degeneration of the lateral columns following a tumor of the pons; once it was noted after the convulsions in unilateral epilepsy; and once it was seen in a case of alcoholism, with an obscure history of convulsions, not examined until some hours after admission, and under observation a very short time—a case where all the tendon reflexes were exaggerated, but for which no cause could be assigned in the short time it could be observed.

In cases where the reflex was thus markedly exaggerated an attempt was made to get the patellar clonus, which was found in five cases. Four of these had disease of the lateral columns, two combined sclerosis, one multiple sclerosis, and one secondary degeneration; the fifth had acute

¹ Quoted by W. R. Gowers, *op. cit.*, p. 31.

² J. M. Charcot, *Localization of Cerebral and Spinal Diseases* [Syd. Soc's translation], 1883, p. 25.

rheumatism with endocarditis, and there was a slight and irregular clonus at times on the left side; the patellar reflex and the deep reflexes of the upper extremity were increased on that side. (See Table V.)

In six cases the patellar reflex was absent on one side: a case of incipient locomotor ataxia, a mild case of multiple neuritis, a case of hemiplegia where it was absent on the paralyzed side, a case of severe crural neuralgia following a fall where it was absent on the affected side, a case of diabetes mellitus with pain and prickly feelings in the legs, and a case of dementia with evidence of lead-poisoning. In nine cases it was diminished on one side: three of these were cases of severe and protracted sciatica where it was less on the affected side, one was a case of hemiplegia where it was most marked on the paralyzed side (this case afterward showed contracture and exaggeration of the reflexes on that side), a case of cerebral meningitis, a case of obscure paraplegia, a case of hypochondriasis, a case of recent hemiplegia where it was diminished on the paralyzed side, and a case of apoplexy. In three cases it was increased on one side: a case of hysterical hemiplegia where it was most marked on the paralyzed side, a case of cerebral syphilis where several deep reflexes were increased on the same side, and a case of paraparesis from being trampled on by a horse. In the case of dementia, where no history could be obtained and no trustworthy examination could be made, it is possible that the lead in the system may have affected the cord or the peripheral nerves. With this exception the difference seems pretty well accounted for except in the case of hypochondriasis. (See Table III.)

In 18 cases out of 231 a reflex was obtained from the periosteum of the tibia—a reflex closely allied to that from the patellar tendon, which Schultz considers an indication of the reflex character of tendon phenomena.¹ In five cases it was but slight, and was not attended with any marked exaggeration of the patellar reflex. In eleven cases the patellar was exaggerated, although not always to such a degree as to be pathological, so that its presence seems to be rather of corroborative value as an indication of exaggeration of the deep reflexes than pathognomonic of disease of the lateral columns. In two cases of hemiplegia it was present on the paralyzed side only.

Second in importance to the patellar reflex, though not in frequency, comes ankle clonus. In 12 cases out of 238 it was found on one or both sides, and in every case there was a definite pathological cause. In four cases it was seen temporarily after a convulsion, once in unilateral epilepsy, on the convulsed side only. The other cases gave clear evidence of disease of the lateral columns: three were cases of combined sclerosis of the posterior and lateral columns, one was a case of multiple sclerosis

¹ R. Schultz, Die Bedeutung der Sehnenreflexe, etc., Deutsches Archiv für kl. Medicin, Feb. 14, 1882.

involving the lateral columns, one was an acute myelitis in the dorsal region with probable descending degeneration, one was a case of secondary degeneration following tumor of the pons, one a unilateral degeneration in old hemiplegia, and one a case of cerebral syphilis with unilateral exaggeration of the deep reflexes. (See Table V.)

I regret to say that no regular test was made of front tap contraction. It was present, of course, in the cases of ankle clonus, and in the case of alcoholism where the patellar reflex was exaggerated. It was tested in a few other cases of exaggerated reflexes, but was not obtained. The wrist and toe clonuses were tested in every case, but never found.

Returning now to the cases in which the patellar reflex was absent, it will be remembered that the larger part of them had some lesion of the reflex arc, but that a considerable percentage presented no evidence of such a lesion. The first nine cases quoted at some detail were cases of cerebral disturbance, usually with the history of alcoholic excess. The tenth case may have some spinal disturbance as a result of his fall; the eleventh was one of those patients whose nervous system is wholly worn out, cases in which at times Berger and Bloch found the reflex absent.¹ The cause of its absence in the twelfth case I cannot determine. Perhaps slight disturbances in children may affect the reflex, since all the children examined lost it under ether, but that is a point which demands further investigation.

It has been admitted, and these observations help to confirm the fact, that the cutaneous reflexes may be lost in cases of disease of the higher centres, that is, of the brain. If the knee phenomenon be really a spinal reflex, why may not disease of the brain in like manner diminish or abolish it?

It has long been known that disease of the pyramidal tract in the brain may be followed by a descending degeneration of the lateral columns in the cord, one of the symptoms of which is an exaggeration of the deep reflexes. This degeneration, according to Schiefferdecker's experiments on dogs, does not begin until the fourteenth day at the earliest,² which agrees with the clinical observations that late contracture in hemiplegia comes on from the fifteenth to the thirtieth day, or even later. In cases of direct hemiplegia, as from a hemorrhage in the middle third of the internal capsule, Charcot asserts that ankle clonus on the paralyzed side precedes the contracture, and that exaggeration of the deep reflexes precedes ankle clonus, the exaggeration being valuable as a premonition of contracture, and occurring, perhaps, before there is actual degeneration in the cord.³ In one case of hemiplegia I found that contracture appeared

¹ Dr. Berger, Ueber Sehnenreflexe, Centralblatt für Nervenhe. etc., Feb. 15, 1879. See also R. Schultz, art. cit.

² Quoted by J. Ross, op. cit., vol. ii. p. 94.

³ J. M. Charcot, op. cit., p. 248 *et seq.*

TABLE V.—Cases showing Ankle and Patellar Clonus.

The same holds true here as with the cases in Table III. See paragraph at head of that table.

Number of observation.	Name.	Disease.	Cutaneous Reflexes.							Deep Reflexes.						
			Plantar.	Cremaster.	Gluteal.	Abdominal.	Ergas-tric.	Erector spinae.	Scapular.							
37	Owen K.	Epilepsy	N.T.	N.T.	N.T.	N.T.	N.T.	N.T.	N.T.	Patellar.	Ankle clonus.	Tibial.	Triceps.	Radial.	Ulnar.	Extensors of wrist.
70	John E.	Uremic convulsions	N.	O.	N.T.	O.	O.	N.T.	N.T.	N. ¹	R. ¹	O.	N.	O.	N.T.	N.T.
202	Robert P.	"	N.T.	N.T.	N.T.	N.T.	N.T.	N.T.	N.T.	N. ¹	R. ¹	O.	N.	O.	N.T.	N.T.
89	Bart. H.	R. unilateral epilepsy [during fits]	R.N.	R.N.	R.N.	R.—	O.	O.	N.T.	N.T.	R. ¹	O.	N.	N.T.	N.T.	N.T.
107	Edwin M.	Multiple sclerosis [left side most affected]	+	N.	O.	N.	N.	R. ¹	R. ¹	+	L.O.	+	N.	R. ¹	R.N.	O.
128	Samuel K.	Tumor of pons	N.	N.	—	O.	O.	O.	O.	R.	clonus	+	+	+	+	+
175	Horace A.	Combined sclerosis [chronic myelitis]	N.	O.	O.	O.	O.	O.	O.	clonus	+	+	N.	R.N.	L.O.	O.
194	Michael F.	"	—	N.	R.O.	O.	O.	O.	O.	+	R. ¹	+	N.	O.	O.	O.
2	Richard L.	Dorsal myelitis	+	—	L.N.	O.	O.	O.	O.	+	L.—	+	+	O.	O.	O.
161	John H.	Chronic myelitis	N.	N.	O.	O.	O.	O.	O.	+	R.—	O.	+	R.—	R.N.	R.—
108	William E.	R. hemiplegia	R.—	N.	O.	N.	R.O.	O.	O.	R.	clonus	O.	N.	N.	N.	N.
186	Nathan W.	Cerebral syphilis	—	N.	N.	R.N.	R.N.	O.	O.	R. ¹	L.O.	O.	—	R.—	R.N.	R.N.

¹ On recovery patellar reflex was only slightly marked, two or three days after any convulsion.

some weeks after a difference in the deep reflexes on the two sides was noted; a second case where a like difference was observed was not under observation long enough to notice whether contracture occurred. That such exaggeration is not always due to structural changes in the lateral columns is shown by the occurrence of ankle clonus after epileptiform convulsions, which was first observed by Hughlings Jackson,¹ and later by Gowers, Beevor,² and others,—a phenomenon which I have noticed in four cases of epileptic and epileptiform convulsions arising from various causes. In such cases there can of course be no wasting of nerve-fibres as in sclerosis, and Jackson explains the phenomenon by the hypothesis of an exhaustion of the nerve-fibres in the lateral columns, thereby perhaps causing a loss of control by the cerebrum, the cerebellar influence being unantagonized, or, what he thinks even more probable, there being also an exhaustion of inhibitory centres in the cord itself.³ When this exhaustion extends down so far as to affect the cells in the lumbar centres, instead of an increase of the reflex, it may be lost entirely, as has been noticed by Westphal⁴ and Gowers,⁵ the latter of whom suggests this theory as an explanation. This will account for the absence of the reflex in Case V.

If new disturbances in the brain which either cut off the cerebral influence by a direct lesion, as in hemiplegia, or by an exhaustion of the conducting fibres, as in convulsions, increase the reflex, why may not an irritative lesion of the motor tract, by increasing the activity of the fibres in the lateral columns, inhibit it? That the lateral columns are excitable, though to a less degree than nerves, has been demonstrated by various observers.⁶ Moreover, the patellar reflex has been occasionally found absent by Westphal on the paralyzed side in hemiplegia.⁷ Why may not the absence of the reflex then, in the cases of cerebral disturbance which I have quoted, be due to the over-activity of the lateral columns from some general irritation in the brain? In most of them spinal symptoms were absent, and the cerebral disturbance was the only discoverable cause of the absence of the reflex. The abuse of alcohol may have been a further factor in the causation, but more observations on similar cases must be made before the point can be determined. The theory, at any rate, seems plausible, as a means of explaining the absence of the reflex.

In further support of this hypothesis I will mention one case a little

¹ J. Hughlings Jackson, On a case of temporary left hemiplegia, with foot-clonus and exaggerated knee-phenomenon, after an epileptiform seizure. *Medical Times and Gazette*, Feb. 12, 1881.

² W. R. Gowers, *On Epilepsy, etc.*, ed. 1881, p. 100.

³ J. Hughlings Jackson, Croonian Lectures on the Evolution and Dissolution of the Nervous System. *Lancet*, April 12, 1884.

⁴ C. Westphal, *art. cit.*

⁵ W. R. Gowers, *On Epilepsy*, p. 101.

⁶ J. M. Charcot, *op. cit.*, p. 235.

⁷ C. Westphal, *art. cit.*

more fully. A man was attacked with sudden paraplegia after excessive coitus, and a diagnosis of acute myelitis was made, which was localized in the lower dorsal and the lumbar regions. Two or three days later, when he entered the hospital, the skin reflexes below the epigastric were absent, and there was no patellar reflex. I did not see the case in its later stages, but Dr. Robert Bell, the house physician at the time, told me that six or eight weeks later he had exaggerated patellar reflex, ankle clonus, and much rigidity and muscular spasm in the legs. Supposing that the upper lumbar region with the dorsal was the part diseased, the irritation of the lateral columns in the lower lumbar region from the acute process may have served to inhibit the reflex at the first, but, after descending degeneration had begun, the influences from above may have been checked in their transmission, the inhibition may thus have been cut off, and the symptoms of a lateral sclerosis have arisen.

This does not, however, seem a satisfactory explanation of the absence of patellar reflex in the cases of coma. This I believe to be due to a paralysis of the reflex centre in the cord. Alcohol and probably ether, and perhaps the poison in uræmia, paralyze the least organized, the most complex, the least automatic, the least perfectly reflex centres first—if they did not, as Jackson says, “death from alcohol would be a very common thing”—hence, in profound alcoholism it is not strange if the centre for the knee phenomenon is paralyzed before the most perfectly reflex centres, those of the respiration and the circulation.

As regards the deep reflexes of the upper extremity Bramwell asserts that they are seldom, if ever, present in health,² and Ross states that they are obtained under circumstances analogous to those in which the patellar reflex is exaggerated.³ In the cases examined, however, some of these reflexes were generally present.

The triceps reflex proved to be the most constant of the deep reflexes, not even excepting the patellar reflex, it being absent in but 41 cases out of 239. It was absent in nine cases of multiple neuritis, two cases of spinal meningitis, two cases of locomotor ataxia where there was but little impairment of co-ordination in the arms, a case of lead paralysis, and a case of diphtheritic paralysis where the legs were chiefly affected. In 22 cases the patellar reflex was present. The other cases were of various sorts, acute and chronic nephritis, neuralgias in different parts, valvular disease of the heart, debility, alcoholism, dyspepsia, etc., so that its absence would seem to be of no particular significance. It was markedly exaggerated in a case of acute dorsal myelitis, during a convulsion in unilateral epilepsy, and in a case of descending degeneration of the cord

¹ J. Hughlings Jackson, Croonian Lectures on the Evolution and Dissolution of the Nervous System. *Lancet*, March 29, 1884.

² B. Bramwell, *op. cit.*, p. 117.

³ J. Ross, *op. cit.*, vol. i. p. 154.

from tumor of the pons. In five cases it differed on the two sides, in three cases of hemiplegia where it was most marked on the paralyzed side, in a case of obscure paraparesis of spinal origin, and in a case of herpes zoster thoracica, where it was absent on the affected side.

The radial reflex was present in 131 cases out of 239, the ulnar in 61, the reflex from the extensor tendons of the wrist in 25. They were absent in too many cases for it to have any special significance, and, although present in many cases of exaggerated patellar reflex, they also occurred in cases of arthritis, hæmatemesis, valvular disease of the heart, etc., where the nervous system was apparently healthy. Exaggeration, though seen in some cases of disease of the lateral columns, was not noticed in every case, and was also noticed without special nervous disorder.

In twelve cases the radial reflex differed on the two sides, in eleven of which there was disease of the brain, cord, or peripheral nerves, clearly or probably more marked on one side than on the other. In a case of marked anæmia both the radial and ulnar reflexes were present to a slight degree on one side only. In six other cases where the ulnar differed there was disease of the brain or cord most pronounced on one side. The reflex from the extensor tendons differed in a case of hemiplegia, and of dorsal myelitis; it was also absent on one side in a case of chronic nephritis, without nervous symptoms. The wrist clonus was never found.

The deep reflexes from the lumbar fascia and the spine of the scapula are of rare occurrence and of insignificant value. The reflex from the spine of the scapula occurred 45 times in 178 cases, that from the lumbar fascia 33 times. A case of unilateral epilepsy was the only one in which they differed.

The last reflex on the list, that from the third costal cartilage, has been observed more especially in phthisis. When the chest is very fat or œdematous it is, of course, obscured, but it was found in 161 cases out of 234. In ten cases where it was increased, six were cases of general exaggeration of the deep reflexes, and four were cases of phthisis. But nine cases of phthisis were examined, in those it was exaggerated in four, of which three were advanced and one incipient; it was normal in four, of which two were advanced, and it was absent in one, which was complicated with acute nephritis and gangrene of the lung. From so small a number of cases of phthisis no deductions can be made, yet I am disposed to believe that the contraction of the pectoral from percussion of the muscle itself—the muscle, perhaps, being in a more irritable condition in phthisis—has been confused with the true reflex from percussion over the third costal cartilage. The costal reflex differed on the two sides in six cases, all of unilateral cerebral disease, tumor, hemiplegia, meningitis, and epilepsy.

The following tables will show the frequency of absence of the various reflexes :—

Cutaneous Reflexes.

	Cases exam'ed.	Present.	Absent.		Cases exam'ed.	Present.	Absent.
Plantar . .	234	217	17	Epigastric . . .	239	97	142
Cremaster .	167	146	21	Erector spinæ . .	178	45	133
Gluteal . .	179	113	66	Scapular . . .	177	15	162
Abdominal .	239	142	97				

Deep Reflexes.

	Cases exam'ed.	Present.	Absent.		Cases exam'ed.	Present.	Absent.
Patellar . .	239	192	47	Ulnar	239	61	178
Tibial . . .	231	18	213	Extensors of wrist	239	25	214
Ankle clonus	238	12	226	Wrist clonus . .	239	0	239
Toe clonus .	239	0	239	Costal	234	161	73
Triceps . .	239	198	41	Lumbar fascia .	178	33	145
Radial . . .	239	131	108	Spine of scapula .	178	45	133

From these observations, then, the following conclusions may be drawn :—

1. Absence of the plantar or cremaster reflex is usually pathological, depending upon a direct lesion of the reflex arc or some cerebral disturbance.

2. Absence of the other cutaneous reflexes is not necessarily pathological.

3. Absence of the patellar reflex may be due to cerebral disturbance, especially in alcoholic subjects.

4. Ankle and patellar clonus are pathological.

5. The deep reflexes of the upper extremity are of frequent occurrence, and have no special pathological significance.

6. The costal reflex is found in the majority of cases without general exaggeration of the reflexes, and with no signs of phthisis, incipient or advanced.

7. When the reflexes differ on the two sides, though it usually signifies some unilateral disease of the nervous system, it is not always pathological.

Finally, these observations have led me to emphasize the value of testing all the reflexes, cutaneous and deep, in the upper extremity as well as in the lower, and on the two sides of the body, in examining patients with nervous diseases.